Interaction of training and diet on metabolism and endurance during exercise in man

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1. Ten untrained young men ingested a carbohydrate-rich diet (65 energy percent (E%) carbohydrate, T-CHO) and ten similar subjects a fat-rich diet (62 E% fat, T-FAT) while endurance training was performed 3–4 times a week for 7 weeks. For another 8th week of training both groups ingested the carbohydrate-rich diet (T-CHO and T-FAT/CHO).

2. Maximal oxygen uptake increased by 11% ($P < 0.05$) in both groups after 7 and 8 weeks. Time to exhaustion at 81% of pre-training maximal oxygen uptake increased significantly from a mean (± S.E.M.) of 35 ± 4 min to 102 ± 5 and 65 ± 7 min in T-CHO and T-FAT, respectively, after 7 weeks ($P < 0.05$, T-CHO *vs.* T-FAT). After 8 weeks, endurance remained unchanged in T-CHO but increased ($P < 0.05$) to 77 ± 9 min in T-FAT/CHO which, however, was still less ($P < 0.05$) than in T-CHO.

3. Muscle glycogen breakdown rate during exercise was halved by endurance training equally in both T-CHO and T-FAT after 7 and 8 weeks, and muscle glycogen stores at exhaustion were not depleted in any group.

4. During exercise after 7 weeks, the respiratory exchange ratio (RER) was unchanged in T-CHO (0·88 ± 0·01) compared with pre-training but decreased ($P < 0.05$) to 0·82 ± 0·02 in T-FAT. After 8 weeks, RER in both T-CHO and T-FAT/CHO was approximately 0·87.

5. During exercise, plasma noradrenaline concentration and heart rate were higher in T-FAT than in T-CHO both at 7 and at 8 weeks.

6. It is concluded that ingesting a fat-rich diet during an endurance training programme is detrimental to improvement in endurance. This is not due to a simple lack of carbohydrate fuel, but rather to suboptimal adaptations that are not remedied by short-term increased carbohydrate availability. Furthermore, the study suggests that the decrease in RER usually seen after training when exercising at the same absolute intensity as before training can be prevented by a carbohydrate-rich diet.

Physical endurance training results in marked changes in muscle metabolism and endurance during exercise. Classical adaptations to training include a switch to increased fat metabolism when exercising on the same absolute intensity as before training (Henriksson, 1977; Koivisto, Hendler, Nadel & Felig, 1982; Kiens, Essen-Gustavsson, Christensen & Saltin, 1993; Coggan, Spina, Kohrt & Holloszy, 1993), less muscle lactate release (Henriksson, 1977; Kiens *et al.* 1993), a blunted hormonal response (Winder, Hagberg, Hickson, Ehsani & McLane, 1978; Koivisto *et al.* 1982; Galbo, 1983) and increased endurance. It is also known that dietary status is of importance for metabolism and performance during exercise. Thus, ingestion of a fat-rich diet for 3–5 days decreases carbohydrate metabolism and performance during exercise in man (Christensen & Hansen, 1939; Bergström, Hermansen, Hultman & Saltin, 1967; Galbo, Holst & Christensen, 1979). However, studies in man (Phinney, Bistrian, Evans, Gervino & Blackburn, 1983; Muoio, Leddy, Horvath, Awad & Pendergast, 1994; Lambert, Speechly, Dennis & Noakes, 1994) and rat (Miller, Bryce & Conlee, 1984; Conlee, Hammer, Winder, Bracken, Nelson & Barnett, 1990; Simi, Sempore, Mayet & Favier, 1991) have also suggested that when a fat-rich diet is ingested for 1–5 weeks, endurance may not be impaired (Phinney *et al.* 1983; Conlee *et al.* 1990) and may even be improved (Miller *et al.* 1984; Simi *et al.* 1991; Muoio *et al.* 1994; Lambert *et al.* 1994). The proposed explanation has been that the fat-rich diet increases muscle fat oxidative capacity and free fatty acid (FFA) concentrations in plasma which more than compensates for the decreased

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carbohydrate stores (Simi *et al.* 1991). Moreover, a recent study in training dogs has shown that a high fat diet (65 energy percent (E%)), consumed for 6 weeks, induced a large increase in mitochondrial volume density measured in the vastus lateralis muscle, as well as a large increase in maximal fat oxidation rate (Taylor, Hoppeler, Kennedy, Valenski, Roberts & Weyand, 1994). Thus, if both training and a fat-rich diet induce adaptations that increase fat oxidative capacity then it might be speculated that combining the two interventions might result in an additive effect and, by inference, in maximal endurance capacity. This has in fact been demonstrated in rats (Simi *et al.* 1991). It could also be hypothesized that if a combination of training and a fat-rich diet was performed then a subsequent short-lasting switch to a carbohydrate-rich diet should create optimal conditions for increased endurance, because a high fat oxidative capacity is combined with large glycogen stores.

The present study has a dual aim. First, we wanted to study if combining an endurance training programme in untrained subjects with a fat-rich diet affected exercise metabolism and endurance performance differently than if a carbohydrate-rich diet was consumed during an identical training programme. Second, we studied exercise metabolism and endurance performance when, after training on a fat-rich diet, the carbohydrate-rich diet was consumed for an additional week of training.

METHODS

Subjects

Twenty untrained healthy male subjects of mean age, 27 years (range, 22–42 years), height, 181 cm (range, 170–193 cm), weight, 82 kg (range, 57–100 kg) and maximal oxygen uptake, 3·7 l min^{-1} (range, 2·9–4·3 l min^{-1}) participated in the study. The fibre type composition in the vastus lateralis muscle averaged 56 ± 4·0% type I, 28 ± 3·0% type IIA and 16 ± 3·0% type IIB fibres. Subjects were fully informed of the nature of the study and the possible risks associated with it before they volunteered to participate, and they gave written consent. The study was approved by the Copenhagen Ethics Committee.

Protocol

The experimental period lasted 8 weeks. Initially subjects were matched in pairs according to their maximal oxygen uptake and the composition of the subject's habitual diet. The subjects were then randomized, one from each pair, into two groups ($n = 10$ in each group). Both groups followed a training programme for 8 weeks and consumed either a carbohydrate-rich (group T-CHO) or a fat-rich (group T-FAT) diet during the first 7 weeks. During the 8th week subjects on the carbohydrate-rich diet remained on this diet, whereas subjects initially on the fat-rich diet switched to the carbohydrate-rich diet (group T-FAT/CHO). Initially and then after 7 and 8 weeks an endurance test to exhaustion was performed on a Krogh bicycle ergometer. Furthermore, maximal oxygen uptake was determined on a Krogh bicycle ergometer before and after 3·5, 6·5 and 7·5 weeks.

Experimental diets

To establish the daily energy intake and composition of the subject's habitual diet, 4-day diet recordings were carried out by all subjects (3 weekdays and 1 weekend day). All food intake and beverages were weighed and recorded and energy intake and composition of the diets were calculated using a computer database (Dankost II; the Danish Catering Centre, Copenhagen, Denmark). In addition, individual energy intakes were determined from the World Health Organization's equation for calculation of energy needs (World Health Organization, 1985).

Two experimental diets were designed, one diet rich in carbohydrate (CHO) and one rich in fat (FAT). The mean energy composition of the carbohydrate-rich diet was 65 E% carbohydrate, 15 E% protein and 20 E% fat. The mean energy composition of the fat-rich diet was 21 E% carbohydrate, 17 E% protein and 62 E% fat. The composition of the two diets was designed to be markedly different in fat and carbohydrate content, and as similar as possible in protein content, fibre content and the ratio between polyunsaturated and saturated fatty acids (P/S ratio) (Table 1). Isocaloric 7-day cycle menus were designed at the subject's individual energy levels and the day-to-day variation was kept as low as possible. On the days of training, the calculated energy expenditure during training was added to the daily energy intake. The intake during the dietary periods was strictly controlled. All food and beverages were prepared accurate to 1 g. Parts of the diets were prepared in the metabolic kitchen and delivered to the subjects. The remaining parts were prepared by the subjects, and all meals were consumed at home. All food intake was registered, and so were all omissions from the prescribed food intake. The subjects weighed themselves every morning and the individual energy level was adjusted according to any change in body weight. Duplicate food collections were performed from all meals in the diets. Food collections were frozen at −20 °C until further analysis. All food collections were analysed individually.

Training

An identical, supervised training programme was followed by both dietary groups. During the first 4 weeks, training was performed 3 times a week and during the last 4 weeks, 4 times a week. Each training session lasted between 60 and 75 min, and exercise intensity, which was carefully controlled, was varied between 50 and 85% of maximal oxygen uptake. Training intensity was adjusted to changes in maximal oxygen uptake measured during the training period. At every training session heart rate was monitored with a heart rate recorder, and pulmonary oxygen uptake was frequently measured.

Experimental protocol

Subjects were asked to refrain from physical activity 2 days prior to the endurance tests. Subjects reported to the laboratory either by car or bus in the morning after a 12 h fast. After 30 min of rest in the supine position, resting metabolic rate and respiratory exchange ratio (RER) were measured. A Venflon catheter was inserted into an antecubital vein and resting blood samples were collected without a cuff. The catheter was flushed with sterile sodium citrate. A needle biopsy was taken with suction from the vastus lateralis muscle under local anaesthesia with lidocaine. After 5 min of rest in the sitting position, another blood sample was taken just prior to exercise. Then an endurance test to exhaustion was performed on a Krogh bicycle ergometer. Subjects commenced working at the selected workload without warm-up.

During exercise, blood samples were taken and oxygen uptake was measured after 5, 15 and then every 15 min and at termination of exercise. Heart rate was recorded continuously. During exercise, subjects drank 200 ml of water every 20 min. Exhaustion was defined as the point at which the pedalling frequency could no longer be maintained at the desired frequency despite verbal encouragement. Immediately after exercise stop, another biopsy was taken with suction from the vastus lateralis muscle through a new incision.

The endurance test to exhaustion was repeated after 7 and 8 weeks, following the same protocol. At the initial test subjects exercised at approximately 81% of maximal oxygen uptake and the same absolute workload was maintained in all three tests. The verbal encouragement was given only by staff from the laboratory, who had no knowledge of the groupings of the subjects. An endurance training session designed as an endurance test was performed exactly 1 week before the 7 week test to obtain identical preconditions for the endurance tests after 7 and 8 weeks of training.

Analyses

Blood sample analysis. Blood glucose and blood lactate were analysed on a glucose and a lactate analyser, respectively (Yellow Springs Instruments, Yellow Springs, OH, USA). Blood glycerol and β-hydroxybutyrate were analysed as described in Wieland (1974) and Bergmeyer, Dermot & Williamson (1974), respectively, adjusted to fluorometric assays. Plasma FFA were measured fluorometrically as described by Kiens *et al.* (1993). Plasma potassium was analysed by flame photometry (Radiometer FLM3, Radiometer, Copenhagen, Denmark). Insulin in plasma was determined using a radioimmunoassay kit kindly donated by Novo-Nordisk (Copenhagen, Denmark) and catecholamines in plasma were determined by a radioenzymatic procedure (Christensen, Vestergaard, Sørensen & Rafaelsen, 1980). Haematocrit was determined in triplicate from microcapillary tubes. Haemoglobin was determined spectrophotometrically by the cyan–methaemoglobin method (Drabkin & Austin, 1935).

Muscle sample analysis. The biopsy samples were frozen in liquid nitrogen within 5–10 s. Before freezing, a section of the samples was cut off, mounted in embedding medium and frozen in isopentane cooled to its freezing point in liquid nitrogen. Both parts of the biopsy were stored at −80 °C until further analysis. Before biochemical analysis, muscle biopsy samples were freeze-dried and dissected free of connective tissue, visible fat and blood using a stereomicroscope. Muscle glycogen concentration was determined as glucose residues after hydrolysis of the muscle sample in 1 M HCl at 100 °C for 2 h (Lowry & Passonneau, 1972). Serial transverse muscle sections were stained for myofibrillar ATPase to identify fibre types (Brooke & Kaiser, 1970). Serial sections were stained for glycogen using the periodic acid–Schiff reagent (PAS) reaction (Pearse, 1968) and the glycogen depletion pattern was evaluated (Gollnick, Piehl & Saltin, 1974).

Oxygen uptake. Pulmonary oxygen uptake at rest and during exercise was determined by collection of expired air in Douglas bags. The volume of air was measured in a Collins bell spirometer (Collins, Braintree, MA, USA) and the fractions of oxygen and carbon dioxide were determined with paramagnetic (Servomex) and infrared (Beckmann LB-2) systems, respectively. Two gas samples with known compositions were used to calibrate both systems regularly.

Heart rate. This was recorded with a PE 3000 Sports Tester (Polar Electro, Finland).

Diet composition. The duplicate food samples were analysed at the National Food Agency of Denmark. Protein content was determined by multiplying the nitrogen content by the factor 6·25. Nitrogen was determined by the Kjeldahl method. Fat content and fatty acid composition were determined by gas chromatography in accordance with Leth (1988). Carbohydrate content was determined by subtraction of ash, protein and fat from total dry matter. Ash content was determined as the residue after heating to 550 °C. Dietary fibre content was determined in accordance with the AOAC method (AOAC, 1985). Energy content was determined in accordance with Lusk (1928).

Statistics

Results are given as means ± S.E.M. if not otherwise stated. For each variable measured, a two-way analysis of variance (ANOVA) with repeated measures for the time factor was performed to test for changes due to training or diet. Between-group differences were tested with an unpaired Student's t test, whereas differences between time points were detected with a Duncan multiple range test. In all cases, a probability (P) of 0·05 was used as the level of significance.

RESULTS

Diet

The subjects' habitual diet was similar in the two groups (Table 1). During the dietary period the actual daily nutrient intake calculated from the dietary diaries returned by the subjects was similar to the planned nutrient intake (Table 1). In both experimental diets, the energy intake was similar, and also similar to the habitual diet. Compared with the subjects' habitual diet, the intake of carbohydrates in the experimental diet was 42% higher on the CHO diet and 53% lower on the FAT diet. The daily intake of fat was 57% lower on the CHO diet and 131% higher on the FAT diet. The CHO and FAT diets, respectively, supplied 17% and 32% more protein compared with the subjects' habitual diet ($P < 0·05$). In group T-FAT/CHO the dietary composition was similar to the CHO-experimental diet (Table 1). Thus, the carbohydrate and fat intakes in the 8th week were significantly different compared with the intake during the previous 7 weeks, but the protein intake was similar.

The analysed fatty acid composition of the diets revealed a distinct difference in dietary fatty acid composition between the two experimental diets, with a markedly higher content of *w-3* fatty acids as well as ω-6 fatty acids in the FAT diet compared with the CHO diet (Table 2). The chemical analysis of the duplicate dietary portions yielded energy content and energy compositions similar to the computer-based calculations (Table 3).

Pulmonary maximal oxygen uptake

Before the onset of the experimental period, maximal oxygen uptake was similar in the two groups and averaged

Table 1. Calculated mean daily intake of energy and nutrients in the subjects' habitual diet and during 7 weeks on an experimental diet

		Habitual diet		Experimental diet	
	Units	T-CHO	T-FAT	T-CHO	T-FAT
Energy	MJ	13·6 ± 0·6	11·9 ± 0·6	14·3 ± 0·4	13·7 ± 0·3
Protein	E%	13·2 ± 0·8	14·3 ± 0·7	14·6 ± 0·1	16·5 ± 0·4*†
	g	105 ± 7	101 ± 8	123 ± 4*	133 ± 3*†
Carbohydrate	E%	48·2 ± 1·8	53·4 ± 2·9	65 ± 0·5*	22 ± 0·2*†
	g	386 ± 28	373 ± 28	546 ± 16*	177 ± 5*†
	g (kg body wt)$^{-1}$	4·7 ± 0·3	5·0 ± 0·4	6·8 ± 0·2*	2·4 ± 0·2*†
Simple sugars	E%	11·0 ± 2·9	10·0 ± 3	7 ± 0·3*	2·2 ± 0·5*†
Dietary fibres	g MJ^{-1}	2·3 ± 0·2	2·6 ± 0·4	4·3 ± 0·02*	2·2 ± 0·02†
Fat	E%	34·3 ± 1·8	39 ± 2·5	20·4 ± 0·4*	62 ± 0·2*†
	g	118 ± 7	94 ± 8	75 ± 2*	217 ± 6*†
Cholesterol	mg MJ^{-1}	31 ± 3·7	29 ± 4·4	26 ± 0·2*	44 ± 5·2*†
Essential FA	E%	4·4 ± 0·4	4·3 ± 0·4	4·0 ± 0·1	11·2 ± 0·1*†
P/S ratio		0·39 ± 0·05	0·43 ± 0·08	0·53 ± 0·01*	0·62 ± 0·01*†

FA, fatty acids; P/S ratio, ratio between polyunsaturated and saturated fatty acids. The intake from the habitual diet was calculated from a 4-day dietary record. The intake on the experimental diet was calculated from dietary diaries kept over the 7 week period. Values are means ± S.E.M. * $P < 0·05$ between the habitual and the experimental diet; † $P < 0·05$ between the two experimental diets.

3·72 ± 0·13 l min^{-1} in T-CHO and 3·58 ± 0·17 l min^{-1} in T-FAT. After 3·5, 6·5 and 7·5 weeks of training, maximal oxygen uptake was 3·93 ± 0·15 ($P < 0·05$ compared with pre-training), 4·09 ± 0·11 and 4·08 ± 0·11 l min^{-1} in T-CHO, and 3·82 ± 0·14 ($P < 0·05$ compared with pre-training), 3·99 ± 0·15 and 4·03 ± 0·08 l min^{-1} in T-FAT, respectively. Thus, at the endurance tests after 7 and 8 weeks, maximal oxygen uptake was significantly increased to the same extent in both T-CHO and in T-FAT.

Resting values

Initially, resting RER values were similar in T-CHO (0·82 ± 0·04) and T-FAT (0·80 ± 0·02) and these values remained at this level after 7 and 8 weeks. Resting heart rate averaged 64 ± 3·0 beats min^{-1} in both groups initially. A similar and significant decrease appeared in both groups during the study, to 59 ± 3·2 and 56 ± 2·0 beats min^{-1} in T-FAT and T-CHO, respectively. Initial resting blood glucose and lactate, plasma FFA and potassium levels were similar in T-CHO and T-FAT and no changes were seen in these parameters during the experimental period (Table 4). Initial resting blood β-hydroxybutyric acid concentration was similar in the two groups and no changes were seen in T-CHO during the study. After 1 week on the fat-rich diet, blood β-hydroxybutyric acid increased from 36 ± 10 to 202 ± 47 μmol l^{-1} ($P < 0·05$) in T-FAT and remained at this level during the following 5 weeks. After 7 weeks, β-hydroxybutyric acid had decreased to 90 ± 30 μmol l^{-1}, which was still significantly higher compared with initial values (Table 4). After 8 weeks it was not different from initial values (Table 4). The plasma glycerol concentration was similar in the two groups initially and after 7 weeks. A slight but significant decrease was seen after 8 weeks in both groups (Table 4). Initial resting insulin concentration was slightly but significantly higher in T-CHO (10·7 ± 1·1 μU ml^{-1}) than in T-FAT (6·7 ± 0·8 μU ml^{-1}) but a similar concentration in the two groups was seen after

Table 2. Daily intake of dietary fatty acids in the experimental diets, based on chemical analysis of the food ingested

	Fatty acid content (mg (100 g dry wt)$^{-1}$)				
	C 18 : 2 ω-6	C 18 : 3 ω-6	C 20 : 5 ω-3	C 22 : 5 ω-3	C 22 : 6 ω-3
CHO diet	480 ± 60	78 ± 10	8 ± 3	2 ± 1	17 ± 10
FAT diet	781 ± 90*	98 ± 40	52 ± 18*	9 ± 1*	76 ± 30*

Fatty acids are given in mg (100 g dry wt)$^{-1}$ in a 10 MJ diet. Values are means ± S.E.M. * $P < 0·05$ between the CHO and the FAT diet. C 18 : 2 ω-6, linoleic acid; C 18 : 3 ω-6, γ-linolenic acid; C 20 : 5 ω-3, eicosapentaenoic acid; C 22 : 5 ω-3, docosapentaenoic acid; C 22 : 6 ω-3, docosahexaenoic acid.

Table 3. Daily intake of energy nutrients in the experimental diets: comparison between computer calculations and chemical analyses

	CHO diet		FAT diet	
	Calculation	Analysis	Calculation	Analysis
Energy (MJ)	10	9·5	10	9·6
Carbohydrates (E%)	64·9	65·7	21·3	22·6
Fat (E%)	20·4	19·0	61·7	60·5
Protein (E%)	14·6	15·4	17·0	17·6
Dietary fibre (g MJ^{-1})	4·3	3·2	2·1	2·4
P/S ratio	0·50	0·68	0·58	0·57

The computer calculations were made using the Dankost II computer database of the Danish Catering Centre.

7 and 8 weeks (Table 4). Initially the concentrations of adrenaline in plasma were similar in the two groups and no changes were seen during the experimental period (Table 4). Similar concentrations of noradrenaline were seen initially and after 7 weeks in both groups. After 8 weeks a small but significant increase appeared in both T-CHO and T-FAT/CHO (Table 4).

Resting blood samples taken in the sitting position just prior to exercise start were generally not different from those taken in the resting supine position. The exception was plasma FFA, where a significantly higher concentration appeared in the sitting position compared with the supine position at the 7 week and 8 week endurance tests in both T-CHO and T-FAT (Fig. 4).

Endurance performance

Initially, endurance time to exhaustion was similar in T-CHO (35·2 ± 4·5 min) and in T-FAT (35·7 ± 3·8 min) (Table 5). When calculated from the 15 min value, subjects exercised at 82 ± 2% and 79 ± 2% of maximal oxygen uptake in T-CHO and T-FAT, respectively. After 7 weeks, endurance time to exhaustion was significantly increased by 191% in T-CHO and by 68% in T-FAT (Table 5). Subjects exercised at 69 ± 1% and 71 ± 2% of maximal oxygen uptake in T- CHO and T-FAT, respectively. After the 8th week no further increase in endurance time to exhaustion was seen in T-CHO whereas endurance time to exhaustion in T-FAT was further increased by 18% ($P < 0·05$) but was still 26% shorter compared with endurance time to exhaustion in T-CHO ($P < 0·05$) (Table 5).

Initial endurance test

During the initial exercise test, RER averaged 0·90 ± 0·02 and 0·94 ± 0·02 in T-FAT and T-CHO, respectively. Heart rate increased progressively in both groups during exercise to 191 ± 6 beats min^{-1} in T-FAT and 181 ± 6 beats min^{-1} in T-CHO at exhaustion. A significant increase was seen in blood lactate to 6·2 ± 0·4 and 5·3 ± 0·6 mmol l^{-1}, in plasma glycerol to 173 ± 27 and 212 ± 29 μmol l^{-1} and in plasma potassium concentrations to 5·1 ± 0·2 and 5·3 ± 0·1 mmol l^{-1} at exhaustion in T-CHO and T-FAT, respectively, whereas blood glucose and plasma β-hydroxybutyric acid concentrations remained unchanged compared with resting values (Table 4) during exercise and at exhaustion (Table 6). Plasma concentrations of FFA decreased significantly in both groups after 15 min of exercise and were still lower ($P < 0·05$) compared with

Table 4. Resting values of glucose and lactate in blood, and of FFA, glycerol, β-hydroxybutyric acid, potassium and hormones in plasma, initially and after 7 and 8 weeks

	0 weeks		7 weeks		8 weeks	
	T-CHO	T-FAT	T-CHO	T-FAT	T-CHO	T-FAT/CHO
Glucose (mmol l^{-1})	4·8 ± 0·2	4·5 ± 0·1	4·5 ± 0·6	4·6 ± 0·1	4·5 ± 0·1	4·5 ± 0·1
Lactate (mmol l^{-1})	0·66 ± 0·1	0·51 ± 0·06	0·66 ± 0·12	0·46 ± 0·08	0·58 ± 0·14	0·49 ± 0·1
FFA (μmol l^{-1})	407 ± 37	477 ± 37	412 ± 46	486 ± 53	352 ± 35	448 ± 36
Glycerol (μmol l^{-1})	83 ± 9	87 ± 14	84 ± 12	88 ± 13	72 ± 10*‡	74 ± 9*‡
β-Hydroxybutyric acid (μmol l^{-1})	20 ± 5	36 ± 10	28 ± 8	90 ± 30*†	25 ± 5	14 ± 5‡
Potassium (mmol l^{-1})	3·95 ± 0·10	3·85 ± 0·07	3·94 ± 0·13	4·07 ± 0·11	4·06 ± 0·19	4·05 ± 0·15
Insulin (μU ml^{-1})	10·7 ± 1·1	6·7 ± 0·8†	8·7 ± 1·6	8·1 ± 1·6	8·3 ± 1·5	9·4 ± 1·3
Adrenaline (ng ml^{-1})	0·06 ± 0·01	0·07 ± 0·01	0·07 ± 0·01	0·10 ± 0·01	0·08 ± 0·01	0·09 ± 0·01
Noradrenaline (ng ml^{-1})	0·24 ± 0·03	0·23 ± 0·04	0·21 ± 0·03	0·29 ± 0·04	0·32 ± 0·04‡	0·42 ± 0·07§

Resting values were measured in the supine position. Values are means ± S.E.M. * $P < 0·05$ between 0 and 7 or 8 weeks. † $P < 0·05$ between groups T-CHO and T-FAT; ‡ $P < 0·05$ between 7 and 8 weeks; § $P = 0·06$ between 0 and 8 weeks.

Table 5. Endurance performance, glycogen in the vastus lateralis muscle before and after exercise, glycogen breakdown and rate of glycogen breakdown, initially and after 7 and 8 weeks training on the experimental diets

	Endurance time (min)	Glycogen before exercise (mmol kg^{-1})	Glycogen after exercise (mmol kg^{-1})	Glycogen breakdown (mmol kg^{-1})	Rate of glycogen breakdown (mmol kg^{-1} min^{-1})
Basal					
T-CHO	35·2 ± 4·5	436 ± 17	226 ± 34§	210 ± 34	6·0 ± 0·6
T-FAT	35·7 ± 3·8	428 ± 14	201 ± 25§	227 ± 25	6·5 ± 0·4
After 7 weeks					
T-CHO	102·4 ± 5·0*†	611 ± 29*†	294 ± 25§	316 ± 41†	3·1 ± 0·4†
T-FAT	65·2 ± 7·2†	511 ± 25	308 ± 49†§	210 ± 34	3·0 ± 0·5†
After 8 weeks					
T-CHO	103·6 ± 7·2*	561 ± 22	291 ± 24§	270 ± 56‡	2·6 ± 0·5
T-FAT/CHO	76·7 ± 8·7‡	738 ± 53*‡	463 ± 59*‡§	275 ± 47	3·6 ± 0·7

All glycogen values are given in mmol (kg dry wt)$^{-1}$. Values are means ± S.E.M. * $P < 0·05$ between T-CHO and T-FAT; † $P < 0·05$ between basal and 7 week values; ‡ $P < 0·05$ between 7 and 8 week values; § $P < 0·05$ between before and after values.

resting values at exhaustion in both T-CHO (305 ± 38 μmol l^{-1}) and T-FAT (341 ± 37 μmol l^{-1}) (Table 6). During exercise the insulin concentration decreased similarly in T-CHO and in T-FAT, from 10·7 ± 1·1 and 6·7 ± 0·8 μU ml^{-1} at rest (Table 4) to 8·2 ± 1·2 and 4·7 ± 1·2 μU ml^{-1} ($P < 0·05$) at exhaustion (Table 6). Adrenaline concentrations increased ($P < 0·05$) similarly in the two groups during exercise, to 0·64 ± 0·13 and 0·86 ± 0·15 ng ml^{-1} in T-CHO and T-FAT, respectively, at exhaustion (Table 6). Noradrenaline concentrations increased significantly during exercise to 3·5 ± 0·6 and 4·4 ± 0·4 ng ml^{-1} in T-CHO and T-FAT, respectively ($P < 0·05$) (Table 6).

Endurance test after 7 weeks

The mean RER values during the 7 week exercise test in T-CHO were unchanged compared with RER values in the initial exercise test, whereas in T-FAT, RER values were significantly lower during the 7 week exercise test compared with those obtained during the first exercise test. The RER values during the 7 week exercise test were significantly lower in T-FAT compared with T-CHO (Fig. 1). Heart rate was significantly higher during exercise in T-FAT compared with T-CHO (Fig. 2) during the 7 week exercise test.

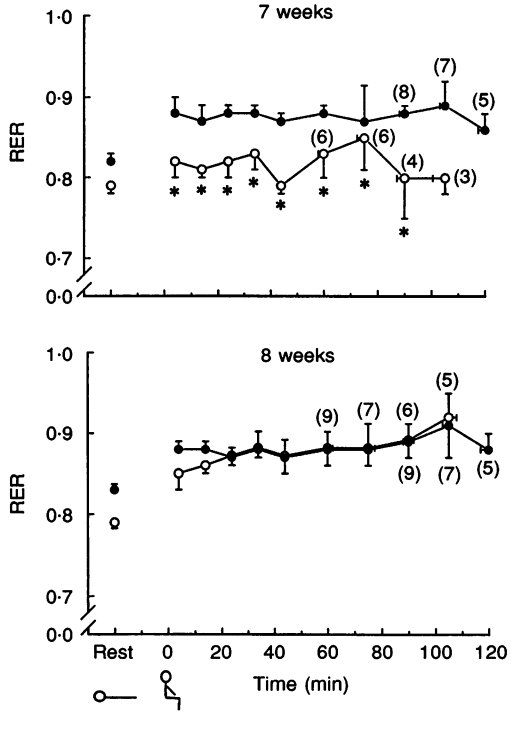

Figure 1. Respiratory exchange ratio (RER)

RER was measured at rest (in the supine and sitting position) and during the endurance exercise test after 7 weeks (top panel) and 8 weeks (bottom panel) of training on either a carbohydrate-rich diet (T-CHO, ●) or a fat-rich diet (T-FAT, ○). During the 8th week, the T-FAT group switched to the carbohydrate-rich diet (bottom panel, T-FAT/CHO, ○). Values are means ± S.E.M. of 10 observations unless otherwise indicated in parentheses in the figure. * $P < 0·05$ compared with T-CHO.

Table 6. Values at exhaustion of glucose and lactate in blood of FFA, glycerol, β-hydroxybutyric acid and hormones in plasma after the three endurance tests, initially (0 weeks) and after 7 and 8 weeks

	0 weeks		7 weeks		8 weeks	
	T-CHO	T-FAT	T-CHO	T-FAT	T-CHO	T-FAT/CHO
Glucose (mmol l^{-1})	4·9 ± 0·3	4·6 ± 0·3	4·0 ± 0·2*	5·4 ± 0·3*†	3·7 ± 0·2*	5·3 ± 0·4*†
Lactate (mmol l^{-1})	6·2 ± 0·4	5·3 ± 0·6	3·6 ± 0·5*	5·2 ± 0·7†	3·7 ± 0·5*	5·5 ± 0·8†
FFA (μmol l^{-1})	305 ± 38	341 ± 37	845 ± 150*	545 ± 101*	840 ± 107*	526 ± 67*†
Glycerol (μmol l^{-1})	173 ± 27	212 ± 29	340 ± 42*	361 ± 50*	308 ± 42*	248 ± 29‡
β-Hydroxybutyric acid (μmol l^{-1})	25 ± 38	28 ± 4·2	58 ± 13*	87 ± 14*	60 ± 9·9*	59 ± 8·4*
Potassium (mmol l^{-1})	5·1 ± 0·2	5·3 ± 0·1	5·3 ± 0·1	5·5 ± 0·2	5·7 ± 0·1	5·4 ± 0·1
Insulin (μU ml^{-1})	8·2 ± 1·2	4·7 ± 1·2†	4·1 ± 1·1*	6·9 ± 1·5	3·2 ± 0·6	6·9 ± 1·3†
Adrenaline (ng ml^{-1})	0·64 ± 0·13	0·86 ± 0·15	1·2 ± 0·26*	0·84 ± 0·17	1·2 ± 0·24*	0·73 ± 0·15
Noradrenaline (ng ml^{-1})	3·5 ± 0·6	4·4 ± 0·4	3·2 ± 0·4	4·9 ± 0·6†	3·8 ± 0·5	5·4 ± 0·8

Values are means ± S.E.M. * $P < 0·05$ between 0 and 7 or 8 weeks. † $P < 0·05$ between T-CHO and T-FAT; ‡ $P < 0·05$ between 7 and 8 weeks.

Blood substrates. Blood glucose concentrations during exercise were not different from resting values in T-FAT. In T-CHO, blood glucose concentrations remained similar to resting values during the first 60 min of exercise, whereafter a continuous decrease ($P < 0·05$) was seen (Fig. 3). At exhaustion, blood glucose was significantly lower in T-CHO than in T-FAT (Table 6). The blood lactate concentrations increased progressively during the first 45 min of exercise in T-FAT to 3·8 ± 0·8 mmol l^{-1} ($P < 0·05$) and remained at this level during the rest of the exercise period. After 30 min of exercise in T-CHO, blood lactate concentrations were increased to 2·5 ± 0·4 mmol l^{-1} ($P < 0·05$) and remained at this level. No differences appeared in blood lactate concentrations between the two groups during the first hour of exercise, whereafter the blood lactate concentration was significantly higher in T-FAT than T-CHO during the remainder of the exercise period. Thus at exhaustion, the blood lactate concentration in T-FAT was significantly higher than in T-CHO (Table 6). The plasma FFA concentration decreased in both groups ($P < 0·05$) after exercise start, whereafter a continuous and similar increase appeared in both groups until exhaustion. At exhaustion, plasma FFA concentrations averaged 545 ± 101 μmol l^{-1} in T-FAT, not different from resting values (Fig. 4 and Table 6). In T-CHO the plasma FFA concentration at exhaustion averaged 845 ± 150 μmol l^{-1}, which was higher compared with resting values ($P < 0·05$) but not different from the values seen in T-FAT at

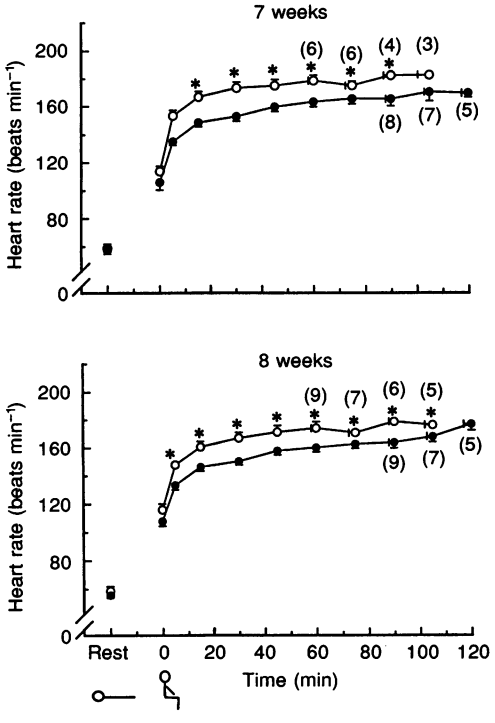

Figure 2. Heart rate

Heart rate was measured at rest (supine and sitting position) and during the endurance exercise test after 7 weeks (top panel) and 8 weeks (bottom panel) of training on either a carbohydrate-rich diet (T- CHO, ●) or a fat-rich diet (T-FAT, ○). During the 8th week, T-FAT switched to the carbohydrate-rich diet (T-FAT/CHO, ○). Values are means ± S.E.M. of 10 observations unless otherwise indicated in the figure. * $P < 0·05$ compared with T-CHO.

exhaustion (Fig. 4 and Table 6). The blood glycerol concentration increased continuously during exercise in both groups but significantly more in T-FAT than in T-CHO (Fig. 5). At exhaustion the blood glycerol concentrations were not different in T-FAT and in T-CHO (Table 6), but significantly higher in both groups than at exhaustion in the initial test.

The concentration of blood β-hydroxybutyric acid remained similar to resting values in T-CHO during exercise, whereas in T-FAT it decreased to 60 ± 17 μmol l^{-1} ($P < 0.05$) during the first 15 min of exercise and remained at this level until exhaustion. Significant differences were not obtained between the groups at exhaustion (Table 6). The plasma potassium concentration increased ($P < 0.05$) to 5.3 ± 0.1 and 5.5 ± 0.2 mmol l^{-1} in T-CHO and T-FAT, respectively, during exercise (Table 6). Differences in exhaustion values between the two groups, or compared with the values at the initial endurance test, did not appear (Table 6).

Hormones. The insulin concentration decreased progressively and similarly during exercise ($P < 0.05$) in T-FAT and in T-CHO. At exhaustion plasma insulin concentrations averaged 4.1 ± 1.1 μU ml^{-1} in T-CHO, not different from T-FAT at exhaustion (Table 6) but lower ($P < 0.05$) compared with the values obtained in T-CHO at the initial test (Table 6). The adrenaline concentration increased similarly and significantly during exercise in T-CHO and in T-FAT, reaching 1.2 ± 0.26 ng ml^{-1} at exhaustion in T-CHO and 0.84 ± 0.17 ng ml^{-1} in T-FAT (Table 6). The noradrenaline concentration increased significantly more in T-FAT than in T-CHO during exercise (Fig. 6) and reached to 4.9 ± 0.6 and 3.2 ± 0.4 ng ml^{-1} ($P < 0.05$) at exhaustion in T-FAT and T-CHO, respectively (Table 6). During exercise the noradrenaline concentration was always higher in T-FAT compared with T-CHO ($P < 0.05$, Fig. 6).

Endurance test after 8 weeks

During the 8th week, the T-FAT group consumed the CHO diet (T-FAT/CHO group). During exercise, RER values in T-FAT/CHO were increased significantly compared with RER values after 7 weeks on the fat-rich diet and were now similar to RER values during exercise in T-CHO (Fig. 1) and to the RER values in the initial test. Heart rate during exercise was still significantly higher in T-FAT/CHO compared with T-CHO (Fig. 2). The blood glucose concentration in T-CHO remained similar to resting values during the first 60 min of exercise followed by a continuous decrease until exercise stop (Fig. 3). In T-FAT/CHO blood glucose increased during the first 30 min of exercise ($P < 0.05$) and remained at this level throughout the experiment (Fig. 3). After 30 min of exercise and until exhaustion, blood glucose concentration was higher in T-FAT/CHO than in T-CHO ($P < 0.05$). At exhaustion the blood glucose concentration was 43% higher in T-FAT/CHO than in T-CHO ($P < 0.05$, Table 6). The blood lactate concentrations in T-FAT/CHO and in T-CHO were similar to the values seen after 7 weeks during exercise and at exhaustion. At exhaustion the blood lactate concentrations were still significantly higher in T-FAT/CHO than in T-CHO (Table 6).

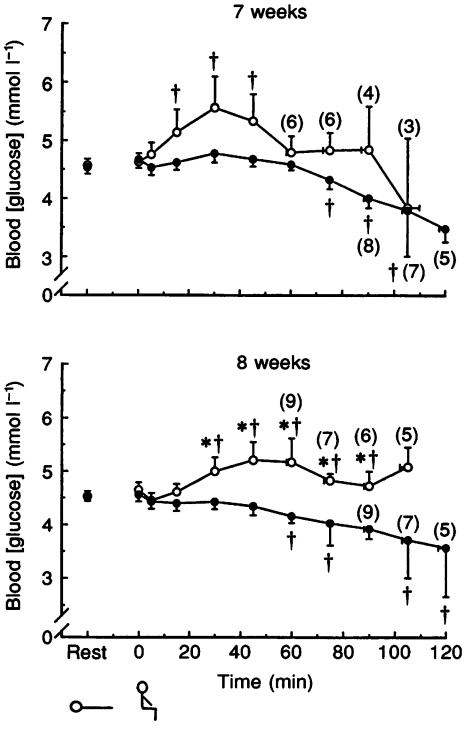

Figure 3. Blood glucose concentrations

Measurements were made at rest (supine and sitting position) and during the endurance exercise test after 7 weeks (top panel) and 8 weeks (bottom panel) of training on either a carbohydrate-rich diet (T-CHO, ●) or a fat-rich diet (T-FAT, ○). During the 8th week, T-FAT switched to the carbohydrate-rich diet (T-FAT/CHO, ○). Values are means ± S.E.M. of 10 observations unless otherwise indicated in the figure. * $P < 0.05$ compared with T-CHO; † $P < 0.05$ compared with resting values.

The plasma FFA concentrations were similar in T-FAT/CHO and T-CHO and similar to the values during exercise after 7 weeks (Fig. 4). At exhaustion the plasma FFA concentrations were 526 ± 67 and 840 ± 107 μmol l^{-1} in T-FAT/CHO and T-CHO, respectively ($P < 0{\cdot}05$) and not different from the 7 week values (Table 6). Plasma glycerol concentrations increased progressively and similarly during exercise, to 248 ± 29 and 308 ± 42 μmol l^{-1} in T-FAT/CHO and T-CHO, respectively, at exhaustion (Fig. 5, Table 6). The plasma glycerol concentration in T-FAT/CHO during exercise was significantly decreased compared with after 7 weeks (Fig. 5) and also lower ($P < 0{\cdot}05$) at exhaustion compared with 7 weeks (Table 6). The plasma β-hydroxybutyric acid concentration increased significantly and similary until exhaustion in T-FAT/CHO (59 ± 8 μmol l^{-1}) and in T-CHO (60 ± 10 μmol l^{-1}). At exhaustion the plasma β-hydroxybutyric acid concentrations were not different compared with the 7 week value (Table 6). The plasma potassium concentrations were similar in T-CHO and T-FAT during exercise and at exhaustion and similar compared with the values after 7 weeks (Table 6).

Hormones. The insulin, adrenaline and noradrenaline responses were similar to the response observed in T-CHO and in T-FAT during exercise after 7 weeks. After 8 weeks the noradrenaline concentration was still significantly higher in T-FAT/CHO than in T-CHO (Fig. 6). At exhaustion the concentrations of insulin were significantly higher in T-FAT/CHO than in T-CHO (Table 6), whereas no significant differences were seen between the groups in plasma adrenaline or noradrenaline concentrations.

Muscle samples

Initially, muscle glycogen values were similar before and after exercise in T-CHO and in T-FAT (Table 5). After 7 weeks the glycogen concentration at rest was increased in T-CHO by 40% ($P < 0{\cdot}05$) and was unchanged in T-FAT, which was significantly less than in T-CHO (Table 5). After 8 weeks muscle glycogen concentrations increased in T-FAT/CHO by 44% ($P < 0{\cdot}05$) compared with after 7 weeks, and was significantly higher compared with T-CHO. No changes appeared in T-CHO compared with the 7 week value (Table 5). No differences in rate of muscle glycogen breakdown were found between the two groups at any of the endurance tests. The glycogen breakdown rate was significantly decreased in both groups after 7 and 8 weeks compared with the initial endurance test (Table 5).

The histochemical analysis revealed that at termination of the 7 week exercise test in T-CHO muscle, glycogen depletion expressed as the percentage of resting values averaged 62% in type I fibres, which was more than in both type II A (35%) ($P < 0{\cdot}05$) and II B fibres (24%) ($P < 0{\cdot}05$). In T-FAT, the muscle glycogen depletion averaged 54% in the type I fibres , which was more than in the type II B fibres (31%, $P < 0{\cdot}05$) but not compared with the type II A fibres (33%). There were no differences in muscle glycogen depletion pattern between the two groups. At termination of exercise in T-CHO after 8 weeks there was a larger depletion of muscle glycogen in the type I fibres (68%) than in the type II A fibres (47%, $P < 0{\cdot}05$), and a larger depletion in type II A than in II B fibres (38%, $P < 0{\cdot}05$). In T-FAT/CHO, the glycogen depletion

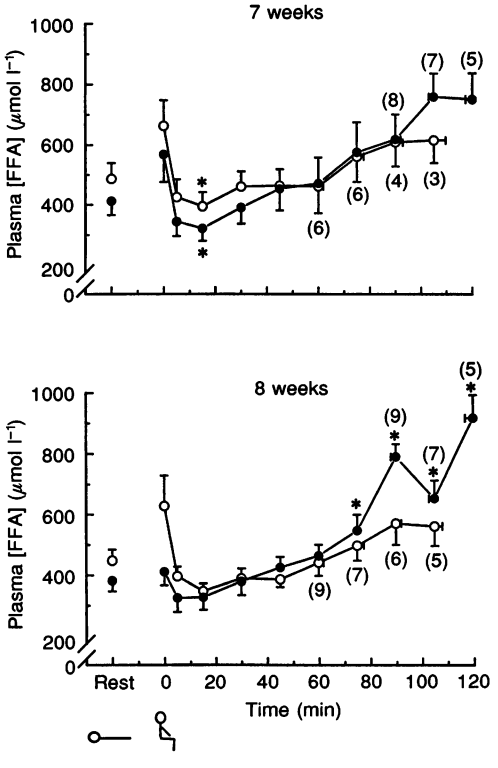

Figure 4. Plasma free fatty acid (FFA) concentrations
Measurements were made at rest (supine and sitting position) and during the endurance exercise test after 7 weeks (top panel) and 8 weeks (bottom panel) of training on either a carbohydrate-rich diet (T-CHO, ●) or a fat-rich diet (T-FAT, ○). During the 8th week, T-FAT switched to the carbohydrate-rich diet (T-FAT/CHO, ○). Values are means ± S.E.M. of 10 observations unless otherwise indicated in the figure. * $P < 0{\cdot}05$ compared with resting values.

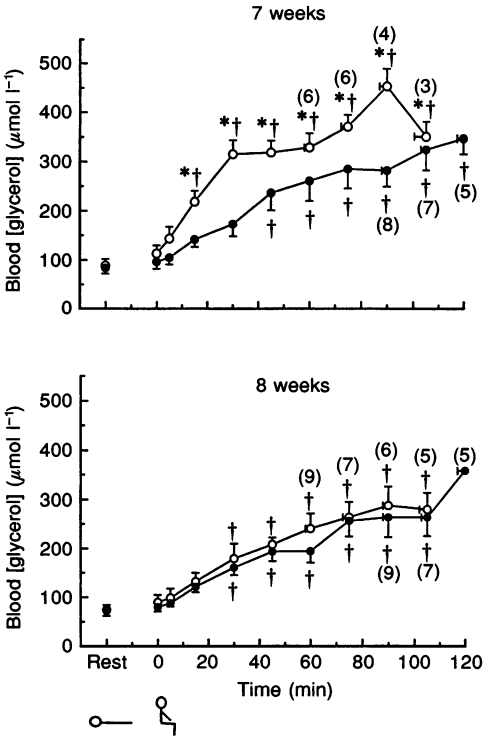

Figure 5. Blood glycerol concentrations
Measurements were carried out at rest (supine and sitting position) and during the endurance exercise test after 7 weeks (top panel) and 8 weeks (bottom panel) of training on either a carbohydrate-rich diet (T-CHO, ●) or a fat-rich diet (T-FAT, ○). During the 8th week, T-FAT switched to the carbohydrate-rich diet (T-FAT/CHO, ○). Values are means ± S.E.M. of 10 observations unless otherwise indicated in the figure. * $P < 0.05$ compared with T-CHO; † $P < 0.05$ compared with resting values.

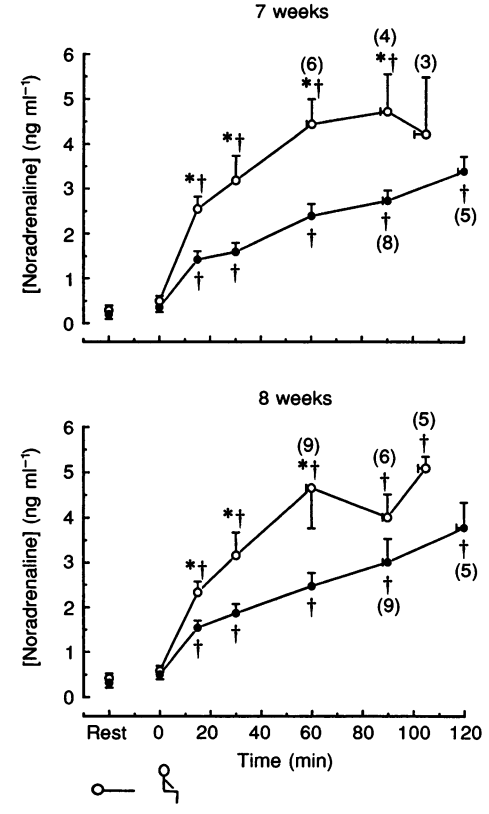

Figure 6. Plasma noradrenaline concentrations
Measurements were made at rest (supine and sitting position) and during the endurance exercise test after 7 weeks (top panel) and 8 weeks (bottom panel) of training on either a carbohydrate-rich diet (T-CHO, ●) or a fat-rich diet (T-FAT, ○). During the 8th week, T-FAT switched to the carbohydrate-rich diet (T-FAT/CHO, ○). Values are means ± S.E.M. of 10 observations unless otherwise indicated in the figure. * $P < 0.05$ compared with T-CHO; † $P < 0.05$ compared with resting values.

averaged 63, 38 and 35% in type I, II A and II B fibres, respectively. However, due to the semiquantitative nature of the PAS staining method used, the conclusion to be drawn from these data is that type I fibres were recruited more than type II fibres and that no major differences in fibre recruitment pattern were found between groups or between tests.

DISCUSSION

In the present study, 7 weeks of aerobic training increased endurance performance markedly more (56%) when a carbohydrate-rich diet was consumed compared with when a fat-rich diet was consumed. Furthermore, when the fat-rich diet was replaced by a carbohydrate-rich diet during an additional week of training, endurance performance was modestly increased, but still significantly lower than in those subjects training on the carbohydrate-rich diet during the entire training period (8 weeks). These findings indicate that training combined with a fat-rich diet induces suboptimal adaptations. This conclusion is dependent upon whether the amount of training was identical in the two groups. That this was the case is supported first of all by the closely supervised training sessions that were identical in the two groups, the absence of additional nonsupervised training and finally by the identical increase in maximal oxygen uptake in the two groups.

It is the prevailing concept that endurance performance after consuming a carbohydrate-rich diet is superior to that when a fat-rich diet is consumed (Christensen & Hansen, 1939; Bergström *et al.* 1967; Galbo *et al.* 1979). In these studies the dietary intervention lasted between 3 and 5 days. However, long-term dietary intervention studies seem to reach different conclusions. Thus, after 4 weeks on a extremely fat-rich diet, endurance performance in five trained cyclists did not change compared with when a balanced diet was consumed (Phinney *et al.* 1983). Recently it was even reported that a fat-rich diet consumed for 2 weeks improved endurance performance during moderate-intensity exercise whereas performance during high-intensity exercise was unchanged (Lambert *et al.* 1994). Also, it has been reported that ingestion of a fat-rich diet for 1 week compared with either a normal or a carbohydrate-rich diet improved both maximal oxygen uptake and endurance (Muoio *et al.* 1994). However, in that study the dietary fat and carbohydrate comprised 38% and 50% of total energy intake, respectively, in the fat-rich diet. Thus, such a diet can hardly be characterized as fat rich. Furthermore, the tests were not performed in randomized order, making interpretation of the results difficult.

In rats, adaptation to a fat-rich diet for 4 (Conlee *et al.* 1990) and 5 weeks (Miller *et al.* 1984) resulted in unchanged or increased endurance performance time, respectively, compared with adaptation to a carbohydrate-rich diet. Thus, dietary intervention studies in both man and rat indicate that a fat-rich diet ingested for 2–5 weeks is not detrimental to endurance performance and may even be beneficial. The proposed mechanism behind a beneficial effect has been adaptations in the oxidative metabolic pathways, such as higher activities of β-hydroxy-acyl-CoA-dehydrogenase (HAD) and citrate synthase (CS) (Miller *et al.* 1984; Simi *et al.* 1991). Such adaptations might then be expected to favour fat combustion to an extent that not only compensates for, but even outweighs, the limited carbohydrate stores.

In the above-mentioned studies, the training status of the subjects was not altered during dietary intervention, and the length of the dietary intervention period was from 1–5 weeks. In contrast, in the present study untrained subjects initiated a 7 week training programme at the same time as the dietary intervention was started. Thus, this study examines the role of diet for the *improvement* in physical performance induced by training. The results clearly indicate that improvement in endurance capacity during 7 weeks' training is markedly better when training is performed on a carbohydrate-rich compared with a fat-rich diet. Our results are in contrast to a study in which rats ran for a longer time after they had been trained on a fat-rich diet compared with training on a carbohydrate-rich diet (Simi *et al.* 1991). However, that was also a study in which the fat-rich diet without training increased exercise performance. One difference between our study and that by Simi *et al.* (1991), in addition to the species difference, is that the endurance test in the rat study lasted for 5–7·5 h after training compared with the 1–2 h in the present study. With an endurance time of 5 h or more the relative exercise intensity is low and not comparable with our study or with generally used endurance tests.

It is not obvious from our data why endurance performance was better in T-CHO than in T-FAT after 7 weeks. Muscle glycogen stores were not depleted in either group and the glycogen depletion pattern was also similar at exhaustion. In addition, at exhaustion blood glucose concentration was higher in T-FAT than in T-CHO (Table 6). This was also the case at exhaustion after 8 weeks, at which time muscle glycogen concentrations in T-FAT/CHO were, furthermore, as high as resting values before initiating training (Table 5). Despite this, endurance performance was still less than in T-CHO. Thus factors other than carbohydrate availability are responsible for the difference in endurance time between the two groups. These observations also indicate that fatigue during prolonged moderately intense exercise does not always seem to be closely related to glycogen depletion, as is usually stated (Christensen & Hansen, 1939; Bergström *et al.* 1967).

It is thought that muscle fatigue originates from changes within the muscle cell rather than from changes in the

neuromuscular transmission (Bigland-Ritchie, Kukulka, Lippold & Woods, 1982; Duchateau & Hainaut, 1985). Studies with single mouse fibres have suggested that muscle fatigue may arise from several mechanisms such as changes of the intracellular milieu causing inhibition of the contractile proteins, reductions of cytosolic calcium concentrations below the maximum for force development, and decreased responsiveness of the contractile proteins to calcium (Renaud & Mainwood, 1985; Westerblad, Lee, Lännergren & Allen, 1991). Recent studies in whole bullfrog semitendinosus muscle also suggest that changes in whole muscle contractility with fatigue may be partially mediated by changes in calcium handling by the cell (Baker, Longuemare, Brandes & Weiner, 1993). The rise in intracellular calcium after depolarization is due to the movement of calcium from sarcoplasmic reticulum to the myoplasm. It has been shown that different types of dietary fat induce changes in the composition of several cellular membranes (Storlien, Jenkins, Chisholm, Pascoe, Khouri & Kraegen, 1991; Pan, Hulbert & Storlien, 1994). The fat-rich diet in the present study had a higher P/S ratio than the CHO diet and in addition contained a much higher proportion of *ω-3* fatty acids (Table 2 and mono-unsaturated acids). Preliminary observations in our laboratory show that changes in human muscle membrane phospholipid composition can be induced in 4 weeks by the fat diet used in the present study (authors' unpublished observations). Thus, if the fat-rich diet induced changes in the lipid composition of the sarcoplasmic reticulum and thereby maybe influenced the transport of calcium to and from the myoplasm, this might be a potential mechanism for the earlier time to exhaustion obtained in the present study in T-FAT compared with T-CHO. This could also explain the maintained shorter endurance time after switching to the carbohydrate-rich diet for 7 days, because diet-induced changes in membranes take more than 7 days.

It is of note that during exercise after 7 weeks, RER values were lower in T-FAT than in T-CHO and the rate of glycogen breakdown was similar in the two groups. This indicates that utilization of blood glucose must have been diminished in T-FAT compared with T-CHO. This was not due to a lower blood glucose concentration in T-FAT than in T-CHO (Fig. 2). Thus, the ability of muscle to utilize blood glucose during exercise must have been less in T-FAT than in T-CHO. Although there is consensus that fat feeding decreases insulin-stimulated glucose transport in rat muscle (Storlien, James, Burleigh, Chisholm & Kraegen, 1986; Rosholt, King & Horton, 1994) apparently related to changes in membrane lipid composition (Storlien *et al.* 1991), there is controversy as to whether fat feeding also decreases exercise-induced glucose transport (Kusunoki *et al.* 1993; Rosholt *et al.* 1994). Therefore, it is difficult to evaluate whether the decrease in glucose utilization in T-FAT compared with T-CHO might be due to diet-induced changes in muscle membrane glucose transport capacity. Alternatively, it could be due to decreased glucose metabolism secondary to increased fat oxidation (Randle, Garland, Hales & Newsholme, 1963; Randle, Newsholme & Garland, 1964) (RER was lower in T-FAT than in T-CHO). However, after 8 weeks RER and rate of glycogen utilization, and consequently rate of glucose utilization, were not significantly different in T-CHO and T-FAT/CHO in spite of a higher plasma glucose concentration in T-FAT/CHO (Fig. 3). Thus, the fact that glucose utilization in T-FAT/CHO was not higher than in T-CHO despite a higher plasma glucose concentration and absence of increased lipid utilization would tend to argue in favour of a diet-induced change in membrane glucose transport capacity.

After 7 weeks of training the heart rate and concentration of noradrenaline were significantly higher during exercise after the fat-rich diet compared with the carbohydrate-rich diet (Figs 2 and 6). A higher level of plasma noradrenaline and heart rate during exercise have also been demonstrated after 5 days on a fat-rich diet compared with 5 days on a carbohydrate-rich diet (Galbo *et al.* 1979; Jansson, Hjemdahl & Kaijser, 1982). This indicates that both in the short and long term, an increased activity in the sympathetic nervous system during exercise is induced by a fat-rich diet compared with a carbohydrate-rich diet. It is of considerable interest, however, that the increased activity in the sympathetic nervous system is maintained after switching from the fat rich diet to the carbohydrate rich diet for 7 days. Thus the maintained increased sympathetic activity in T-FAT/CHO cannot be ascribed to acute carbohydrate deficiency, since muscle glycogen concentrations were high and the RER value was similar to that in the CHO group (Fig. 1). The high activity in the sympathetic nervous system during exercise after switching to the carbohydrate-rich diet in T-FAT therefore suggests that training while consuming a fat-rich diet induces long-term adaptations in the body that elicit an augmented increase in sympathetic nervous activity during exercise. This might be considered an indicator of higher cardiovascular demands in the fat-fed group. If so, such increased demands may be contributing to the poorer endurance in the group on the fat-rich diet.

In earlier studies it has been shown that the RER during submaximal exercise is decreased after a period of endurance training (Koivisto *et al.* 1982; Kiens *et al.* 1993; Coggan *et al.* 1993), indicating a shift towards a greater reliance on fat than carbohydrates as fuels. Thus, the prevailing concept from training studies is that the lower RER values and the higher fat combustion after training compared with before, when working on the same absolute workload, is a consequence of the training stimuli. However, in the present study RER values after 7 weeks of training on the carbohydrate-rich diet were not different

from pre-training RER values when working on the same absolute workload. One could argue that a factor contributing to this lack of change in RER was probably that carbohydrate intake was increased from 48 to 65 E% (Table 1). Recent data from our laboratory, however, indicate that this shift in RER is also not seen, even though subjects before the first pre-training endurance test adapt to the carbohydrate-rich diet for 3 days (authors' unpublished observations). Thus our findings indicate that the shift towards a higher fat utilization during exercise as stated in earlier training studies, in which carbohydrate intake during training was not controlled, may be due to inadequate amounts of dietary carbohydrate relative to training demand. This view is supported by the present findings of a significantly lower RER value during exercise in T-FAT than in T-CHO after 7 weeks training. Furthermore, when T-FAT switched to the carbohydrate diet for 7 days, the RER value during exercise increased and was similar to the value in T-CHO and the pre-training RER values.

In conclusion, the present study has shown that a combination of training and a fat-rich diet did not reveal an additive effect on physical performance in man. In contrast, endurance performance of untrained men is markedly improved when endurance training for 7 weeks is performed while ingesting a carbohydrate-rich rather than a fat-rich diet. Thus, the present data give no support to short-term studies claiming that training on a fat-rich diet does not impair, and may even improve, performance. Furthermore, there was only a minor benefit when switching to the carbohydrate-rich diet since differences in endurance (although diminished) persisted when both groups for the final 8th week of training ingested the same carbohydrate-rich diet. It appears as if adaptation to a fat-rich diet induces suboptimal adaptations that are not remedied by suddenly increased carbohydrate availability.

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